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NATURALLY-OCCURRING AND EXPERIMENTAL THIAMIN DEFICIENCY IN CATS RECEIVING COMMERCIAL CAT FOOD

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Introduction

THIAMIN DEFICIENCY produces characteristic clinical and pathological manifestations in the cat (9). Known as Chastek paralysis in the fox, thiamin deficiency also occurs naturally in mink, horse (5), and possibly, the ruminant (11). In the cat, fox, and mink, the deficiency is apparently a result of inadequate levels in the food (9), while in the horse, ingestion of certain plants containing a thiaminase apparently causes the disease (5, 15). The cause in ruminants is uncertain (4).

The following report describes five clinical cases of thiamin deficiency in cats, the results of some thiamin analyses of commercial cat food available in Canada, and a small feeding experiment.

Case #1

In June 1967 a three-year-old spayed female domestic shorthair cat was presented to the Western College of Veterinary Medicine (WCVM) for examination. The owner had noted incoordination that evening and an inability on the part of the cat to jump onto a chair without falling. Except for ataxia, no other abnormalities were noted. Therapy consisted of vitamin B complex¹, chloromycetin² and prednisolone³ parenterally administered. Coordination improved and the animal was discharged seven days later.

In August 1969 the cat was readmitted as

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¹Vijecta, Austin Laboratories Ltd., Paris, Ontario.
²Animycetin, Stevenson, Turner & Boyce, London, Ontario.

³Delta-Cortef, Upjohn Co., Kalamazoo, Michigan.

an evening emergency because of convulsions and dyspnea. The cat had several clonic convulsions of short duration (30 seconds) which could be precipitated by handling. Between convulsions the cat remained in lateral or sternal recumbency.

On physical examination, the cat was obese and had a pendulous abdomen. The pupils were dilated and unresponsive to light although the ocular fundus was normal. Supporting and postural reflexes were absent. Marked ventroflexion of the head occurred during the convulsions and while suspended by the hindlegs. The rectal temperature was normal to subnormal.

The results of cerebrospinal fluid analysis, complete blood count, and skull radiographs were normal. Therapy consisted of supportive fluids, and hot water bottles were used to maintain body temperature. Progressive deterioration was followed by death on August 19.

Permission for a necropsy was obtained.

Subsequent to the cat's death, it was learned that the owner had bought a case of chickentype cat food about three weeks before the onset of signs.

Necropsy Results (Case #1)

Gross and microscopic examination of the brain revealed lesions consistent with a diagnosis of thiamin deficiency: bilateral hemorrhage, edema, neuronal necrosis and capillary activation in the inferior colliculi and lateral geniculate bodies.

Case #2

A seven-year-old castrated male domestic shorthair cat was presented to the WCVM in March 1969 for examination. The owner's complaint was that for four days the cat had been straining, unable to support its hindquarters, and had not been observed to eat or urinate during that period of time. The owner feared a urethral obstruction because the cat had been treated for this in November 1966. The cat

also had a history of a chronic bilateral otitis externa. The diet was a commercial liver/beef-type of cat food; a case had been bought about two weeks previously.

On physical examination, the cat was in poor condition, depressed and was estimated to have been about 6% dehydrated. The urinary bladder was not distended. The cat evidenced marked incoordination, ventroflexion of the head when suspended by the hindlegs, and dilated, unresponsive pupils (Figure 1). Placing reflexes were variable in the front legs and

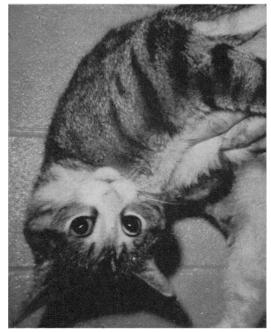


FIGURE 1. Case #2. Note the ventroflexion of the head and the dilated pupils.

absent in the hind legs and the righting reflex was subnormal. The temperature was 99.6°F and the pulse was 200 per minute. The left ear contained an accumulation of cerumen and the tympanic membrane appeared to have been scarred. Radiographs of the tympanic bullae, a complete blood count, and the blood urea nitrogen (BUN) concentration were normal. Urinalysis indicated the presence of cystitis.

A diagnosis of thiamin deficiency was made and therapy consisted of oral alimentation and 1 cc of a multiple B vitamin preparation⁴ b.i.d. Gradual improvement occurred and the cat was discharged 12 days after admittance with a mild degree of ataxia. A multiple vitamin preparation⁵ was dispensed to add to the food.

Case #3

In the same month the owner of case #2 submitted a four-year-old male domestic shorthair cat to the WCVM. The complaint was that the cat was suffering an apparent disorientation. The neurological manifestations were similar to case #2, i.e. ventroflexion of the head when suspended, ataxia, subnormal righting and placing reflexes, and mydriasis. The tail was held erect, as if being used for balancing. The rectal temperature was 102°F and the pulse was 140 per minute. A complete blood count was normal. Therapy consisted of the administration of 75 mg thiamin in a multiple vitamin preparation, given twice a day. The cat responded rapidly and was discharged six days later with a moderate amount of ataxia.

Cases #2 and #3 were seen again about three weeks after admission and were improved, but still had a mild degree of ataxia. Conversation with the owner six months later indicated that no ataxia was present, but that case #2 was reluctant to jump onto the furniture.

Case #4

In May 1969 a four and one-half-year-old spayed female domestic shorthair cat was presented to the WCVM because of anorexia of ten days' duration. The cat had also been vomiting, was "off balance", and had been weak for the previous two days. The cat was being fed a commercial preparation of chicken or liver. The owner bought two weeks' supply of food at a time. On examination, the cat was in good physical condition. It had a tremor, however, ventroflexion of head, and variable placing reflexes. The pupils were markedly dilated. The cat walked with its tail erect. Blood thiamin concentration was 8 μgm/100 ml blood.

Therapy consisted of daily injections of a multiple B vitamin preparation⁶ and brewers yeast tablets. Progressive improvement resulted and the animal was discharged after six days. A multiple vitamin preparation⁷ was dispensed.

Case #5

A 14-year-old male domestic shorthair cat was presented to the WCVM in May 1969. It manifested posterior paralysis, weakness, anorexia, and occasional vomiting. The anorexia had been present for five days and the staggering, one day.

The diet was a commercial chicken-type preparation which was bought in quantities of six to eight tins.

⁴Vijecta, Austin Laboratories Ltd., Paris, Ontario (25 mg thiamin per cc).

⁵Vitamycin, Pitman-Moore, Don Mills, Ontario.

⁶Vitamaster, J. Webster Laboratories, Toronto, Ontario

⁷Vitamycin, Pitman-Moore, Don Mills, Ontario.

On physical examination, the cat was thin and had a rectal temperature of 99.4°F. Marked incoordination, ventroflexion of head, tremors, whining, slow righting reflexes, and mydriasis were present. Complete blood count, urinalysis, BUN concentration and fecal examination were within normal limits.

Therapy consisted of a multiple B-vitamin preparation. Rapid improvement occurred and the cat was discharged after five days with a multiple vitamin preparation to be added to the food. Two weeks later the cat was reexamined and exhibited no residual signs.

DETERMINATION OF THIAMIN CONTENT OF CAT FOOD

The cans of cat food were opened and samples weighing about 10 gm (exact weights were recorded) were removed by taking a core of food from the center of the can with a curved spatula. Papain (0.5 gm) and 10 ml distilled water were added to each sample in 125 ml Erlenmeyer flasks, followed by mixing. Digestion was allowed to proceed either for 4 hrs at 50°C or overnight at 37.5°C. Two ml 1 N hydrochloric acid and 18 ml 0.1 N HCl then were added, and each flask was autoclaved for 30 minutes. After addition of 20 ml 0.2% diastase in 2 N sodium acetate, digestion was carried out for 4 hrs at 50°C, followed by filtration using #1 Whatman filter paper. Thiamin was determined as thiochrome by fluorescence measurement of samples and standards on a spectrofluorometer⁸ (10).

No attempt was made to sample the market in a statistically random manner. Unopened cans either were purchased in stores or ob-

⁸Farrand Spectrofluorometer Model 104243, Farrand Optical Co., Inc., Mount Vernon, New York.

tained from cat owners' case lots from which food had been fed to cats which developed clinical signs (Table I).

FEEDING EXPERIMENT

Because each of the five cats described had been fed the same brand of commercial cat food as a complete diet, and because of the frequency of low thiamin concentrations in many of the cat foods tested (Table I), a feeding experiment was initiated.

Three cats were used. One cat, an eightmonth-old female (#12), was fed liver-beef-type cat food (Brand E, Table I); the second, also an eight-month-old female (#14), was fed fish-type cat food, but died from unrelated causes after the second week; the third, a two-year-old male (#13), was fed fish-type cat food (Brand E, Table I). The tins of cat food fed to cat #12 were from the same case of food fed to clinical cases #2 and #3; the food fed to cat #13 was made by the same manufacturer but purchased separately, and contained more thiamin.

The experiment was begun in April 1969. Blood samples were obtained weekly and analyzed for total lactate, pyruvate, and thiamin using the methods of Barker and Summerson (2) and Friedemann and Haugen (6), as modified by Huckabee (8), and Myint and Hauser (10). Results are in Table II.

The animals were observed daily; appetite and coordination were checked specifically. Partial anorexia was noted by the 13th day of the experiment in both cats. This persisted for eight days, after which normal appetite had returned to cat #13 while cat #12 still had partial anorexia, had become thin, and was exhibiting posterior incoordination. On days 25

TABLE I
THIAMIN CONCENTRATION AND PH OF SOME COMMERCIALLY AVAILABLE
CAT FOODS

Brand†	Type or Flavor	pН	Thiamin µg/100 gm‡	Source	
A	Dietary		80	Store	
\mathbf{B} .	Not stated		20	Store	
С	Liver		130	Store	
D	Tuna		90	Store	
E	Liver-beef		10	Store	
E*	Liver-beef	6.3	10	Cases 2 and 3	
E*	Liver-beef	6.3	30	Cases 2 and 3	
\mathbf{E}	Chicken		0	Case 5	
Ē	Fish	6.4	290	Store	
E**	Fish		240	Store	
$\overline{\mathbf{E}}$	Chicken		20	Case 5	

^{*}Fed to cat #12 in feeding trial

^{**}Fed to cat #13 in feeding trial

[†]Each letter represents one can tested

[‡]Wet weight basis

and 27 cat #12 vomited. It was ataxic on day 35, but there was no ventroflexion of the head and the pupils were normal.

After 40 days cat #12 was very ataxic, had markedly dilated pupils, and its tail was rigidly extended caudally. Throughout this time, cat

#13 remained apparently normal.

Ventroflexion of the head of cat #12 was observed when the cat was suspended by the hindquarters on day 44. Righting reflexes were subnormal. At that time, 12 mg thiamin hydrochloride9 was administered intramuscularly. The next day the pupils were not dilated and reacted normally to light, but slight ventroflexion of the head and subnormal righting reflexes were still present. Twelve mg thiamin hydrochloride was given b.i.d. After two days on treatment, improvement was noted in the righting reflexes and there was no ventroflexion of the head. Thiamin therapy continued until the 50th day, at which time euthanasia was performed. Progressive improvement was noted throughout this time, although the cat was consistently unable to land properly after jumping from a table to the floor.

Necropsy Results (Experimental Cat #12)

There were focal areas of edema, hemorrhage, and gliosis in the periventricular gray matter in the brain and brain stem, and some dilation of the venules. Aside from some congestion of hepatic sinusoids, no other lesions were observed.

DISCUSSION

The thiamin requirement of the cat is believed to be approximately 0.4 mg per day, or, because of its role in carbohydrate metabolism, 0.1 mg per 50 kcal consumed per day (13). The acceptable concentration of thiamin in a diet thus can be based upon either the total daily food consumption or total daily caloric intake. The daily caloric requirement for maintenance of the cat is 60 kcal/kg body weight (7). Since caloric intake is an important determinant of total food consumption (14), the caloric content of any food would be assumed to affect the concentration of thiamin required.¹⁰

To calculate the thiamin requirement for maintenance of a cat weighing 3 kg (6.6 lbs), the caloric content of the food must be known or estimated; a reasonable estimate for tinned, moist, meat-type cat food is approximately 500 kcal per 15 oz or 16 oz tin (1). A 3 kg cat requires, for maintenance, 180 kcal/day (3 × 60), or approximately 36% (180/500) of a can of food per day; many cats may eat more than this, however. Based on the 180 kcal/day required, a 3 kg cat requires 0.36 mg (360 µg) thiamin per day (0.1 mg per 50 kcal). If a 3 kg cat consumed an entire 15 oz (approximately 427 gm) tin of food daily, the thiamin concentration should be approximately 84 µg per 100 gm on a wet weight basis. If such a cat consumed 36% of a 15 oz tin per day, as may be possible if the estimate of 500 kcal per tin is reasonably accurate, approximately 235 µg thiamin per 100 gm is needed in the food. Thus an acceptable approximate range of thiamin concentration in tinned cat food designed to be a complete diet would be 84–235 μ g/100 gm for a 3 kg cat.

The thiamin concentrations shown in Table I demonstrate the variability of thiamin concentrations among several brands of cat food. The food fed to clinical cases 2, 3, and 5 is inadequate in thiamin content, based on the calculations above. The food fed to cases 1 and 4 could not be obtained for analysis. The difference in thiamin concentrations of brand E tins fed in the experiment to cats #12 and #13 was reflected in both the clinical course and blood pyruvate concentrations (Table II).

Thiamin can be destroyed by processing, by thiaminases present in certain fish species, by the presence of sulphite (3), and perhaps in other ways (see reviews in 9 and 13). As a result, adequacy should be assured by post-processing analysis rather than by a pre-processing supplementation alone.

Since anorexia is a clinical feature of thiamin deficiency, it is of interest to note that the brain lesions of thiamin deficiency in rats have been shown to occur only in the deficient animals and not in control rats receiving thiamin plus diets isocaloric with those of the thiamin deficient rats (12). This finding further indicates the apparent specificity of the lesions of

thiamin deficiency.

Prevention of thiamin deficiency by owners would probably be accomplished by varying the brands of cat food used, supplementing the diet with rich natural sources of thiamin or with commercially available supplements, and by purchasing different lots of cat food to avoid the possibility of obtaining a series of deficient tins. The clinical management of cases of the disease should include, in addition to the administration of thiamin, due regard to the secondary problems associated with anorexia and metabolic acidosis.

⁹Vi-B₁ Solution, Haver-Lockhart Laboratories, Shawnee, Kansas.

¹⁰Growing cats appear to require a substantial proportion of their calories as protein; adults require somewhat less as protein (7).

THIAMIN DEFICIENCY

TABLE II

THIAMIN, TOTAL LACTATE, AND PYRUVATE IN THE BLOOD OF EXPERIMENTAL CATS.

DIETS FED ARE LISTED IN TABLE I

Blood Thiamin (µg/100 ml)										
Cat	Day	0	7	14	21	28	35	42	49	
#12		32	28	32	12	NA*	NA	NA	NA	
#13		32	19	25	15	NA	NA	NA	NA	
#1 4		28	34	19	_		_	-	_	
	Mean	31								
			Ble	ood Total	Lactate (n	nM/1.)				
#12		1.53	2.66	3.64	NA `	ŇÁ	NA	NA	NA	
#13		2.15	1.44	1.28	NA	NA	NA	NA	NA	
#14		1.00	7.28	2.04	-		-			
	Mean	1.56								
				Blood Pyr	uvate (mN	I/1.)				
#12		0.16	0.19	0.37°	$0.4\hat{6}$	0.26	0.22	0.42	0.08	
#13		0.22	0.10	0.15	0.10	0.12	0.14	0.08	0.20	
#1 4		0.15	0.22	0.20	_			_		
	Mean	0.18								

^{*}NA = not analyzed

Addendum: Since the preparation of this report, another case, a domestic shorthair, was admitted with a history of eating the same brand of liver-beef food purchased in quantity. Anorexia, mydriasis, ataxia, and spinal hyperesthesia were present, and the blood thiamin concentration was 8.0 µg/100 ml. Treatment with multiple B vitamins and dietary incorporation of pork, cereals and brewers yeast was followed after ten days by disappearance of all signs.

SUMMARY

Five cases of thiamin deficiency in cats are described. All the cats were being fed the same brand of commercial cat food. Chemical analyses and a small feeding experiment confirmed the thiamin inadequacy of the food.

RÉSUMÉ

Description de cinq cas de déficience en thiamine chez le chat. Tous les animaux recevaient une nourriture pour chats de même marque de fabrication. Des analyses chimiques et une expérimentation limitée confirmèrent la pauvreté de la nourriture en thiamine.

REFERENCES

- Anon. Basic guide to canine nutrition. White Plains, New York: Gaines Dog Research Center. 1965.
- BARKER, S. B. and W. H. SUMMERSON. The colorimetric determination of lactic acid in biological material. J. biol. Chem. 138: 535. 1941.
- 3. Bhagat, B. and M. F. Lockett. The effect of sulphite in solid diets on the growth of rats. Fd. Cosmet. Tox. 2: 1. 1964.
- 4. EDWIN, E. E., G. LEWIS and R. ALLCROFT.

- Cerebrocortical necrosis: a hypothesis for the possible role of thiaminases in its pathogenesis. Vet. Rec. 83: 176. 1968.
- Evans, E. T. R., W. C. Evans and H. E. Roberts. Studies on bracken poisoning in the horse. Part II. Br. vet. J. 107. 399. 1951.
- FRIEDEMANN, T. E. and G. E. HAUGEN. Pyruvic acid. 2. The determination of keto acids in blood and urine. J. biol. Chem. 147: 415. 1943.
- GREAVES, J. P. Protein and calorie requirements of the feline. In Canine and Feline Nutritional Requirements. O. Graham-Jones, Editor. Oxford: Pergamon Press. 1965.
- Editor. Oxford: Pergamon Press. 1965.

 8. HUCKABEE, W. E. Control of concentration gradients of pyruvate and lactate across cell membranes in blood. J. appl. Physiol. 9: 163. 1956.
- Jubb, K. V., L. Z. Saunders and H. V. Coates. Thiamine deficiency encephalopathy in cats. J. comp. Path. 66: 217. 1956.
- MYINT, T. and H. B. HOUSER. The determination of thiamine in small amounts of whole blood and serum by a simplified thiochrome method. Clin. Chem. 11: 617. 1965.
- Pill, A. H. Evidence of thiamine deficiency in calves affected with cerebrocortical necrosis. Vet. Rec. 81: 178. 1967.
- ROBERTSON, D. M., S. M. WASAN and D. B. SKINNER. Ultrastructural features of early brain stem lesions of thiamine-deficient rats. Am. J. Path. 52: 1081. 1968.
- Scott, P. P. Minerals and vitamins in feline nutrition. In Canine and Feline Nutritional Requirements. O. Graham-Jones, Editor. Oxford: Pergamon Press. 1965.
- 14. Stevenson, J. A. F. Mechanisms in the control of food and water intake. Ann. N.Y. Acad. Sci. 157: 1069. 1969.
- WESWIG, P. H., A. M. FREED and J. R. HAAG. Antithiamine activity of plant materials. J. biol. Chem. 165: 737. 1946.